

**Division 1:  
Prehospital  
Environment**

**Section 8: Assessment and Management of Shock**



**Introduction**

The student must have successfully completed the following sections prior to participating in this section:

Section 1. Roles and Responsibilities

Section 2. EMS Systems

Section 3. Medical/Legal Considerations

Section 4. Medical Terminology

Section 5. EMS Communications

Section 6. General Patient Assessment and Initial Management

Section 7. Airway Management and Ventilation

The understanding and management of shock or cellular oxygenation is the essence of all patient care. If the treatment is done inadequately or too late the patient will die immediately from cardiac failure or in a few days to a few weeks from other organ failure such as lung, kidney or liver, or may survive but without brain function.

The problem is not mystic either in etiology, pathophysiology or management, but the EMT must understand it and make this understanding work for him, not against him. For example: At times the best treatment may be airway, fluids, Pneumatic antishock garment (PASG), and rapid transportation to a hospital. At other times there may be intensive on scene care.

**Objectives**

At the completion of this section, the student will be able to:

- 1.8.1 Define shock based on aerobic and anaerobic metabolism.
- 1.8.2 Discuss the prevention of anaerobic metabolism.
- 1.8.3 Discuss red blood cell oxygenation in the lungs based on alveolar O<sub>2</sub> levels and transportation across the alveolar capillary wall.
- 1.8.4 Discuss tissue oxygenation based on tissue perfusion and release of oxygen.
- 1.8.5 Discuss the role played by respiration, inadequate ventilation in the management of shock.
- 1.8.6 Describe perfusion and the mechanisms of improvement of cardiac output based on the strength and rate of contractions.
- 1.8.7 Discuss the fluid component of the cardiovascular system and the relationship between the volume of the fluid and the size of the container.
- 1.8.8 Discuss systemic vascular resistance, the relationship of diastolic pressure to the SVR and the effect of diastolic pressure on coronary circulation.
- 1.8.9 Discuss the container size in its relationship to the fluid volume and the effect on blood returning to the heart.
- 1.8.10 Discuss body fluids based on total body water, intracellular fluid, and extracellular fluid.
- 1.8.11 Identify the significant anions and cations in the body.
- 1.8.12 Describe the role of protein.
- 1.8.13 Discuss osmosis. Define semi-permeable membranes, and discuss their function.
- 1.8.14 Define isotonic fluids, hypotonic fluids, and hypertonic fluids
- 1.8.15 Define and discuss diffusion.
- 1.8.16 Define active transport.

- 1.8.17 Describe the mechanisms of concentration of electrolytes.
- 1.8.18 Define acid-base balance.
- 1.8.19 Discuss acid-base balance based on hydrogenion concentration, pH, buffer systems.  
Define and discuss the following:
  - a. Respiratory acidosis.
  - b. Respiratory alkalosis.
  - c. Metabolic acidosis.
  - d. Metabolic alkalosis.
- 1.8.21 Describe the mechanism of the body response to perfusion change.
- 1.8.22 Identify the role of the baroreceptor.
- 1.8.23 Describe how the actions of the baroreceptor affect blood pressure and perfusion.
- 1.8.24 Describe compensated shock.
- 1.8.25 Describe uncompensated shock, both cardiac and peripheral effects.
- 1.8.26 Discuss the assessment of the patient's perfusion status, based on physical observations within the primary survey, including pulse, skin, temperature, capillary refill.  
Discuss the relationship of the neurological exam to assessment of hypoperfusion and oxygenation.
- 8.28 Describe the information provided by the following in physical examination: pulse, blood pressure, diastolic pressure, systolic pressure, skin color, appearance, temperature, and respiration.
- 8.29 Discuss management of a shocky patient. Include red cell oxygenation, tissue ischemic sensitivity, IV fluids, the pneumatic antishock garment.
- 1.8.30 Describe the beneficial and detrimental effects of the pneumatic antishock garment.  
Describe the indication and contraindications for the pneumatic antishock garment.
- 1.8.32 Discuss fluid replacement, the types of fluid that are available, the benefits and detrimental effects of each.
- 1.8.33 Discuss how fluid replacement is monitored and controlled.
- 1.8.34 Discuss the routes of fluid replacement and the advantages and disadvantages of each.
- S1.1.35 Demonstrate in order of priority the steps of shock resuscitation.
- S1.1.36 Demonstrate the use of the pneumatic antishock garment (PASG)
- S1.1.37 Demonstrate the proper technique to insert an intravenous catheter.



Definition

- A. Parameters of measurement are not an adequate definition
  - 1. Blood pressure, pulse or respiration
- B. Inadequate cellular oxygenation produces anaerobic metabolism Anaerobic metabolism equals shock
  - 1. Aerobic metabolism is dependent upon
    - a. RBC oxygenation
      - i. Alveolar  $O_2$  levels
        - (a) Airway
        - (b) Ventilation
        - (c)  $FiO_2$
    - b. Transport of RBC, transport across alveolar/capillary wall. This is dependent on no edema to reduce passage, presence of RBC in capillary and ventilation of the alveolus
  - 2. Tissue oxygenation
    - a. Adequate number of RBC
    - b. Adequate tissue perfusion
    - c. Adequate release of oxygen

Physiology of Perfusion

- A. Heart output or effectiveness is dependent on three components
  - 1. Strength of contractions
  - 2. Rate of contractions
  - 3. Volume of blood
- B. Fluid
  - 1. Fluid volume
  - 2. Systemic vascular resistance (SVR) is another name for peripheral resistance. The diastolic pressure is an estimate of SVR. The amount of back pressure during diastole also determines the flow of blood through the coronary circulation
- C. Container
  - 1. Fluid volume and container size
    - a. 5 liter container and 5 liters of fluid = full container
    - b. 5 liter container and 3 liters of fluid = partially full container
    - c. 3 liter container and 3 liters of fluid = full container
    - d. 7 liter container and 5 liters of fluid = partially full container
- D. Cellular Physiology
  - 1. Body fluids
    - a. Total body water 60% adult weight divided into two components
    - b. Intracellular fluid inside the cell membrane 40% body weight
    - c. Extracellular fluid outside the cell 20% body weight
  - 2. Electrolytes ions
    - a. Cations sodium ( $Na^+$ ) Potassium ( $K^+$ ) Calcium ( $Ca^{++}$ )
    - b. Anions chloride ( $Cl^-$ ) bicarbonate ( $HCO_3^-$ )
  - 3. Protein
    - a. Albumin
    - b. Plasma
    - c. Lymph

## INSTRUCTOR'S NOTES

Prevention of anaerobic

Pump.

Volume/size of container.

Standard normovolemic  
adult.

Hypovolemic adult. Blood  
loss from acute hemorrhage  
or severe dehydration.

TBW.

ICF. water & electrolytes.

ECF water & electrolytes.

Salt.

Positive charge.

Negative charge.

- d. Other
- 4. Osmosis
  - a. Semipermeable membrane
    - i. Water, freely interchangeable on both sides
    - ii. Electrolytes cannot actively cross to other side.
    - iii. Water crosses to equalize the concentration—higher concentration pulls fluid from lower concentrations
  - b. Isotonic fluids. Osmotic pressure is equal to normal body fluid
  - c. Hypotonic. Osmotic pressure less than that of normal body fluids
  - d. Hypertonic. Osmotic pressure greater than that of normal body fluids
- 5. Diffusion. Solute molecules can cross membranes but at a slower rate than water
- 6. Active transport
  - a. Larger molecules can be moved across membranes
  - b. Molecules can move toward higher concentrations
  - c. Energy is required
  - d. Faster than diffusion
- 7. Concentration of electrolytes
  - a. Water follows sodium
  - b. Potassium ( $K^+$ ) is the chief intracellular ion
  - c. Sodium ( $Na^+$ ) is the chief extracellular ion
  - d. Changes in ion concentrations affect skeletal and cardiac muscle cells ability to function ( $K^+$ ,  $Ca^+$ ,  $Mg^+$ )
- 8. Acid-base balance
  - a. Definition: concentration of hydrogen ions in body fluids  $[H^+]$
  - b. pH expression of  $[H^+]$ 
    - i.  $pH = 7.35$  to  $7.45$   $pH$  below  $7.35$  is acidosis (excess  $[H^+]$ )
    - ii.  $pH$  above  $7.45$  is alkalosis (low  $[H^+]$ )
  - c. Buffer system offsets minor changes in pH
    - i. Carbonate buffer system can absorb  $20/1$   $H^+$  without a noticeable change in pH
    - ii. Change is as follows:  
 $CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$ —this process can proceed in either direction  $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons CO_2 + H_2O$
  - d. Respiratory system pH changes not managed by the buffers
    - i. If the lungs cannot release  $CO_2$ , the shift is toward  $H^+ + HCO_3^-$  or an excess  $[H^+]$
    - ii. If too much  $CO_2$  is released from the lungs the  $[H^+]$  is below normal. When the lungs are responsible for this change it is called respiratory alkalosis or acidosis
  - e. Kidneys are slowest but most efficient managers of pH changes.
    - i. If the etiology is built up at  $[H^+]$  because of increased production of  $H^+$  in the cells, it is known as metabolic acidosis



## INSTRUCTOR'S NOTES

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Osmotic pressure. <

Small molecules cross much faster than do larger (NaCl vs. protein).

Acidosis.

Alkalosis.

Result of anaerobic metabolism.

- ii. If the etiology is excessive loss of  $[H^+]$  from the kidney or GI tract, the result is called metabolic alkalosis
- iii. The kidneys can retain or release  $H_2CO_3$  to balance  $[H^+]$  and influence pH

**Pathophysiology****A. Mechanism**

- 1. Baroreceptors
  - a. Detection
  - b. Transmission
- 2. Response to baroreceptors discharge
  - a. Sympathetic nervous system
    - i. Cardiac effects
      - (a) Increased strength of contractions
      - (b) Increased rate of contractions
    - ii. Peripheral effects
      - (a) Arteriolar constriction
      - (b) Decreased container size
      - (c) Increased peripheral resistance
  - b. Adrenal
    - i. Cardiac
    - ii. Peripheral

**B. Compensated shock**

- 1. Increased strength of contractions
- 2. Increased rate of contractions increase in heart rate
- 3. Increased systemic peripheral resistance, increase in diastolic pressure

**C. Uncompensated shock**

- 1. Pump unable to maintain pressure
  - a. Cardiac effects
    - i. Decreased volume
      - (a) Increased strength of contractions
      - (b) Increased rate contractions
    - ii. Decreased myocardial strength
      - (a) Ischemia
        - (i) Decreased RBC oxygenation
        - (ii) Decreased cardiac perfusion
          - (aa) Coronary blood flow
          - (bb) Diastolic pressure
      - (b) Necrosis of myocardium
        - (i) Infarction
        - (ii) Increased size of infarction secondary to decreased perfusion
- 2. Peripheral effects
  - a. Peripheral vascular pooling
  - b. Cellular changes
    - i. Decreased perfusion means decreased oxygenation of tissue cells

## INSTRUCTOR'S NOTES

Carotid artery.

Low pressure.

To brain.

Norepinephrine.

Epinephrine.

Maintenance of systolic B/P.

Not detectable by field  
evaluation.

Pulse.

Narrowing of pulse pressure.

Decrease in systolic and  
diastolic pressure.

Availability of fluid.

- ii Decreased available  $O_2$  changes aerobic metabolism to anaerobic metabolism

**Assessment****A. Primary survey****1. Airway/ventilation adequacy****2. Circulation****a. Pulse**

- i. Rate
- ii. Character
- iii. Location

**b. Skin**

- i. Color
- ii. Appearance
  - (a) Pale
  - (b) Cyanotic
  - (c) Mottled
- iii. Temperature
  - (a) Warm
  - (b) Cool
- iv. Moist/dry

**c. Capillary refill**

- i. Less than 2 seconds
- ii. More than 2 seconds

**3. Disability**

- a. Confusion, disorientation, agitation, may result from decreased cerebral perfusion and decreased oxygenation of brain cells
- b. Mini-neurological survey

**B. Secondary survey****1. Blood pressure****2. Head to toe****C. Monitoring****1. Pulse**

- a. Normal rate even with 10% to 15% volume deficit
- b. Detection altered by peripheral resistance
  - i. Radial pulse absent: systolic pressure  $<80$
  - ii. Femoral pulse absent: systolic pressure  $<70$
  - iii. Carotid systolic pressure  $>60$
- c. Character of pulse may reflect circulatory status

**2. Diastolic pressure**

- a. Increase initially with increased peripheral resistance
- b. Coronary blood flow
- c. Normal with 15% to 20% volume deficit

**3. Systolic pressure**

- a. Normal with 20% volume deficit
- b. Reflects cardiac contractility

## **INSTRUCTOR'S NOTES**

Good perfusion.  
Inadequate perfusion.

As to cause of shock.

4. Skin
  - a. Color—RBC oxygenation
  - b. Appearance
    - i. Pale—decreased perfusion (ischemia)
    - ii. Cyanotic—pooling
    - iii. Mottled—combination most common
  - c. Temperature
    - i. Perfusion
    - ii. Heat retention
5. Respiration. Acidosis produces increased  $[H^+]$ . The body responds by increasing the respiratory rate to remove  $CO_2$ . Removal of  $CO_2$  reduces  $[H^+]$   $[H^+] + HCO_3^- \rightarrow H_2O + CO_2$

## Management

- A. RBC oxygenation
  1. Airway
  2. Breathing
  3. Transport
- B. Tissue ischemic-sensitivity
  1. GI, liver, kidneys—45 to 60 minutes (without  $O_2$ )
  2. Muscle, skin—two to three hours
- C. Pneumatic anti-shock garment
  1. Container size reduction and/or increased vascular resistance beneath device
  2. Perfusion of very ischemia-sensitive tissues
  3. Effects
    - a. Capitanace vessels
      - i. Shifts pooled blood
      - ii. Increased upper body blood
      - iii. Part of blood volume may be translocated to upper portion of body
      - iv. Increased pressure of blood returning to the heart
    - b. Peripheral resistance increased
      - i. Decreased blood flow to ischemic resistance tissues
      - ii. No change in peripheral resistance of ishemic sensitive tissues
    - c. Diaphragmatic excursion
      - i. 50% movement reduction
      - ii. Increased intrathoracic pressure
      - iii.  $Pa.O_2, Ph, PaCO_2$  not compromised
    - d. Tissue pressure—Indirectly transmitted through the skin subcutaneously, and muscle tissue surrounding muscle
    - e. Rigid external stabilization
      - i. Pelvic fracture reduction/stabilization
      - ii. Femur stabilization
4. Clinical consequences
  - a. Increased upper body blood volume

## **INSTRUCTOR'S NOTES**

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Ischemic

Length of time an organ can remain viable.

PASG

At time of printing the exact mechanisms of action are under investigation.

Theoretical.

Selective.

Muscles and skin of legs.

Deep.

Traction splint superior without shock.

- i. Increased blood pressure
  - (a) Increased cardiac output
  - (b) Distended neck veins
- ii. Increased pulmonary blood volume
  - (a) Increased cardiac output with intra-pulmonary pressure
- iii. Increased lower body peripheral resistance
  - (a) Increased flow to upper body organs
  - (b) Decreased perfusion to lower body organs if systolic pressure is less than 80 mm Hg
- iv. Increased tissue pressure
  - (a) Reduction of intra-abdominal hemorrhage
  - (b) Reduction of pelvic fracture hemorrhage
  - (c) Reduction of lower abdomen retroperitoneal hemorrhage
- v. Decreased diaphragmatic excursion
  - (a) Observation for ventilation and oxygenation inadequacy
  - (b) Increased intrathoracic pressure
    - (i) Increased cardiac output with CPR
    - (ii) Increased cardiac output with respiration

## 5. Clinical applications

- a. Shock resuscitation
  - i. Increased blood return
  - ii. Increased vascular resistance
  - iii. Increased blood pressure
  - iv. Decreased heart rate
  - v. Increased perfusion of organs in upper half of body
- b. Hemorrhage control
  - i. Intra-abdominal
    - (a) Aorta
    - (b) Liver, spleen
    - (c) Retroperitoneal
    - (d) Pelvic
  - ii. Lower extremities
    - (a) Skin, muscle
    - (b) Femur
- c. Fracture stabilization
  - i. Rigid external immobilization
  - ii. Femur
  - iii. Pelvis
- d. Cardiac arrest
  - i. Increased cardiac output
    - (a) Increased blood return
    - (b) Increased intrathoracic pressure
    - (c) Increased pulmonary blood volume
  - ii. Increased carotid blood flow
    - (a) Increased cardiac output



## INSTRUCTOR'S NOTES

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Lower extremities, abdomen.

Controversial at time of  
printing.

2x.

- (b) Selective increased peripheral resistance
  - iii. Increased cerebral blood flow
    - (a) 3% to 10%
    - (b) Increased but still deficient
- D. Fluid replacement
  - 1. Whole blood
  - 2. Crystalloid
    - a. Ringer's lactate
      - i. Isotonic
      - ii. Buffer when metabolized
    - b. normal saline
      - i. Isotonic
      - ii. No buffer
    - c. Volume expansion
      - i. Immediately effective
      - ii. 2/3 lost to intraatrial space in one hour
    - d. 2 to 3 liters maximum
  - 3. Glucose
    - a. Immediate volume expansion
    - b. Rapid loss
    - c. Resultant free H<sub>2</sub>O increase
  - 4. Plasma
    - a. Volume expansion not presently a prehospital fluid
    - b. Storage and cost reduce prehospital usefulness
  - 5. Dextran
    - a. Volume expansion
    - b. Type and cross match abnormality
    - c. Bleeding abnormalities
- E. Rate of replacement
  - 1. Monitoring parameters
    - a. Pulse
    - b. Blood pressure
    - c. Skin—color, temperature, capillary refill time
- F. Routes of fluid replacement
  - 1. Rational
    - a. Replacement as rapid as possible may be necessary
    - b. Rate of administration determined by
      - i. Length of catheter
      - ii. Internal diameter of catheter
        - (a) Small increase in radius equals large increase in area
        - (b) Area inside of lumen determines the rate of flow
        - (c) Maximum rate
        - (d) 22 gauge preferred for medical patient
        - (e) 16–14 gauge preferred for trauma patient
      - iii. Size of vein has no relationship to flow

## INSTRUCTOR'S NOTES

Most desirable because of O<sub>2</sub> carrying; however, is not a fluid which is available for prehospital use.

Because of rapid loss of crystalloid from the cardiovascular space the patient should be transported to the hospital as rapidly as possible.

5 to 15 min. as glucose metabolized.

Not a prehospital technique

Inversely.  
Directly.

Are dependent upon size (gauge).

- iv. Central line has no prehospital use
- 2. Peripheral
  - a. Advantages
    - i. Rapid identification
    - ii. Minimal equipment
    - iii. Rapid insertion
    - iv. Can be accomplished while other things going on
    - v. Maintenance good
    - vi. Easily accessible
  - b. Disadvantages
    - i. In severe volume depletion is difficult to locate vessels
    - ii. Collapse and roll easily
- G. Steps in shock management for severely injured patients
  - 1. Extrication
  - 2. Pneumatic antishock garment application
  - 3. Stabilization
  - 4. Load for transportation
  - 5. Administration of IV fluids (en route to hospital)
    - a. Ambulance en route to hospital
      - i. Preliminary steps for IV
        - (a) Tourniquet
        - (b) Vein identified
        - (c) Tape torn
        - (d) IV set up
        - (e) Skin prepped
        - (f) Ambulance stopped
        - (g) Insert catheter
        - (h) Needle taped down
        - (i) Ambulance en route again
        - (j) Fluid rate adjusted

## INSTRUCTOR'S NOTES

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Time required to start IV before rolling ambulance toward hospital includes setup time for the administration set, tearing the tape, etc. If all of this is accomplished while the ambulance is en route & stopped only long enough to insert the needle the patient will arrive at the hospital several minutes faster.

